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Longitudinal Relation of Community-Level Income Inequality and Mortality in Costa Rica

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Abstract

The controversy regarding the direct relationship between income distribution and health remains unresolved. Empirical evidence has often failed to advance our understanding because in the countries studied there was limited ability to distinguish hypotheses. This study examines the relation between inequality and mortality in the context of Costa Rica. Costa Rica's unique social and political structure makes confounding through resource and political channels less likely, thus any effects would work predominantly through direct psychosocial channels. Using mortality data extracted from the Vital Statistics Registry, we evaluate the longitudinal relations between income inequality and cause-specific mortality in Costa Rica from 1989–2005. For those aged 15–60, results indicate that there is a significant adverse relation between increases in lagged inequality and mortality from liver disease, and marginal adverse relations with mortality from diabetes and suicide. For those aged 60 and over, there is a limited evidence of a relation between inequality and health. These results suggest increases in inequality may impact health behavior of the working aged population in Costa Rica.

Keywords

Costa Rica; Inequalities; Mortality; Socio-Economic; Small-Area Study

INTRODUCTION

The relation between income inequality and health has been the topic of hundreds of articles, and the results of existing studies are inconsistent. When studies do find that income inequality is associated with poorer health, the interpretation of that association has been the subject of much controversy (Deaton and Lubotsky, 2003; Lynch et al., 2004a; Subramanian and Kawachi, 2004; Wilkinson and Pickett, 2006). Nonetheless, interest in the topic remains high—especially because income inequality continues to grow in most countries (Ferreira and Ravallion, 2008).

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¹This work was conducted at University of California, Berkeley.

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There are three key issues in the research to date that make it difficult to take steps towards resolving the controversy of how to interpret relations between income inequality and health based on empirical evidence: 1) inability to distinguish between broad theories due to the settings in which research has been conducted, 2) inability to clarify proximal mechanisms due to the outcomes examined, and 3) inability to reasonably interpret observed associations due to lack of consideration of appropriate time intervals between income inequality and health outcomes. These issues are elaborated below.

The controversy around income inequality hinges on different theories about why we might observe associations between income inequality and health outcomes (Marmot and Wilkinson, 2001). Three general groups of theories have been proposed to explain the associations between health and income distribution: psychosocial, resource allocation, and neomaterial. The proposed psychosocial theory is the only one of the three whereby inequality *itself* has a direct negative impact on health. The psychosocial theory posits that the perception of social hierarchy causes frustration or invidious comparisons leading to chronic stress and at the same time may lead to diminishing social capital within a community (Kawachi et al., 2004). The increased stress coupled with the limited availability of social support could lead to increased substitution towards poor health behaviors as a coping mechanism (Brown et al., 2006). Alternatively, many argue that income inequality may affect health through its impact on local resource allocation. For example, areas with high inequality may also have lower investment in local resources due to divergent preferences and local taxation laws, and the consequent lack of resources affects health rather than inequality itself (Lynch et al., 2004a, b). Finally, the neomaterial view of the inequality and health relation contends that deep societal and contextual processes generate both inequality and poor health. Under this interpretation, the effects of income inequality on health are contingent on the distribution of other social resources in the society thus, income inequality proxies a combination and accumulation of other negative exposures, lack of individual resources, and systematic underinvestment in health and social infrastructure (Lynch et al., 2000). Disparities among racial groups and entrenched discrimination are often cited examples of the neomaterial mechanism.

The most consistent evidence of a positive association between income inequality and mortality has been found in the US -- a setting in which the three theories cannot be adequately distinguished due to confounding by race and history. For instance, Deaton & Lubotsky (2003) argued that the effects of racial composition confound studies that have found that mortality rates are positively correlated with income inequality across the cities and states in the US. Specifically they found that the fraction of the population that is black was positively correlated with average white incomes, and negatively correlated with average black incomes. Between-group income inequality is therefore higher where the fraction black is higher, as is income inequality in general. Areas where the fraction black was historically higher have also been areas with underinvestment in many dimensions. Thus, the history of slavery and the taxation laws regarding distribution of local goods make it difficult to dispute a role for the resource allocation or neomaterial interpretations in the US, but a psychosocial interpretation cannot be ruled out. In addition and beyond the long-term effects of slavery, the overall heterogeneity within US in terms of ethnicity, race and even language make it one of the most heterogeneous populations in the world and add to the difficulty of disentangling mechanisms.

While studies outside the United States have also found a positive association between inequality and poor health, these studies have some important limitations. For instance, a recent study found that relative deprivation, a related construct to income inequality, was associated with mortality in South Africa for each race group (Salti, 2010). Again, this setting's recent history of Apartheid makes it an exceptional example. Although, the authors

examine associations separately by race, there could be spillover effects from one race to another, as was found in the United States (Deaton and Lubotsky, 2003). Still other studies have found relations between inequality and health in less heterogeneous settings (e.g., Western Europe (Hildebrand and Van Kerm, 2009) and Latin American (Subramanian et al., 2003), but examined self-reported health, which may be more susceptible to bias (Deaton, 2003). Thus, there is a need for research on inequality and objective health measures in racially homogenous settings in order to distinguish between the theories called upon to explain inequality and health relations.

Further, examining a range of health outcomes in relation to inequality may help elucidate more proximate mechanisms. For example, certain causes of death are directly related to behavior and lifestyle choices. If we see increases in these causes of death in areas with increasing inequality, we expect that the relation may be mediated through changes in health behaviors. If health behaviors are implicated in the pathway, then future research could focus on appropriate policy interventions. Unfortunately, most careful longitudinal analyses have focused on all-cause mortality and/or self-reported health. To the extent that cause-specific mortality can illuminate patterns, it should be examined alongside these other outcomes.

It is also important for research on inequality and health outcomes to consider the relation over plausible time intervals. While relations of inequality with health behaviors might be expected over a relatively short time interval, relations with mortality would only be plausible with a time lag of many years. Consequently, careful attention should be paid to the use of appropriate time lags for the inequality exposure given the mortality is often the outcome under study (Glymour, 2008). Most studies have examined contemporaneous inequality and health outcomes (most often self-reported health) and thus have not examined the relation in a way that is consistent with plausible timing of effect. The few studies that considered lags suggest the strongest relation between income inequality and all-cause mortality occurs at lags of 10–15 years (Blakely et al., 2002; Milyo and Mellor, 2003; Subramanian and Kawachi, 2004).

Attempting to address some of the issues outlined above, we examined the longitudinal relation between income inequality and mortality from different causes in Costa Rica. To remedy the first problem, we selected Costa Rica because the theories presented above are more likely to be distinguishable due to several institutional characteristics. First, Costa Rica has a highly homogeneous population. Using the sum of the ethnic, linguistic, and religious fractionalizations measures created by Alesina et al. (Alesina et al., 2003), Costa Rica ranks in the bottom decile in the world in terms of combined ethnic, linguistic and religious heterogeneity. As a frame of reference, South Africa ranks in the 90th percentile, the US ranks about the 70th percentile, China ranks in the 30th percentile, and Costa Rica ranks in the 10th percentile. Clearly, ethnic differences are less likely to confound compared to other well-studied cases. Second, Costa Rica has a highly centralized political system which allocates health service, education, and most local resources, (Mesa-Lago, 2000) making the possibility of *systematic* local underinvestment in unequal areas unlikely. In addition, Costa Rica also has a nationally run universal health insurance regime with an independent budget, making political cycles less significant in the delivery of health services. Thus, in Costa Rica any association between inequality and health would most plausibly reflect the psychosocial pathway, as opposed to the neomaterial or resource allocation pathway. Finally, Costa Rica has had increasing income inequality at both national and local in the past 20 years, making it possible to examine changes over a relatively recent period. In addition, these changes are not due to increasing poverty but rather due to increases in income at the higher end of the income distribution. These patterns are presented below in Figure 1.²

To clarify the issue of proximate mechanisms, we examined all-cause mortality in conjunction with cause-specific mortality for 14 causes and consider age carefully. If a stronger association is found between inequality and mortality from causes related to lifestyle or psychological conditions, we infer that behavior was a key mediator. We also considered two age groups separately because inequality may affect working age populations differently than the elderly through labor market conditions.

To advance the third issue regarding the timing, we consider both *changes* in contemporaneous and 10 year lagged inequality.

METHODS

Data

The data for these analyses combine three data sources obtained from the Central American Population Center (CCP), a research center at the University of Costa Rica. We construct a synthetic panel data set from microdata from the Costa Rican Household Surveys for Multiple Purposes (CRHS), the Vital Statistics Registry of Costa Rica, and intercensal population estimates. The outcome of interest, number of deaths, was extracted from the Vital Statistics Registry of Costa Rica. These data include information on the canton of residence at the time of death, date of death, age at death, gender, and the International Classification of Disease (ICD) codes for the cause of death for each recorded death. Accordingly, these data can be used to calculate death rates for age, gender, year of death, and canton by cause of death with a denominator for each year, canton, age, and gender cell from intercensal interpolations. For most of independent covariate, individual records from the CRHS are aggregated into cells defined by year and canton. The death rates and the CRHS data are merged at the canton & year cell level.

Using the Vital Statistics Registry data, an all-cause of death category was created by summing the total number of death records for each year, canton, five-year age, and gender cell. CCP further classified cause of death into 24 categories by grouping ICD8, ICD9 & ICD10 codes together based on the underlying cause of death. Thus, individual records were also aggregated into cells defined by year, canton, five-year age groups, and gender by cause of death. Fourteen of 24 causes of death groupings were further examined for the detailed cause of death analysis. Ten causes of death categories were not included because these categories have either too few deaths (diarrhea, malnutrition, HIV, and congenital disease) were associated with childbearing (perinatal mortality and maternal mortality), or their underlying cause was not clearly specified (other infections, other cancers, other accidents, and other residual mortality). The fourteen causes examined account for approximately 63% percent of all deaths for those aged 15–59 and 69% of deaths for those aged 60 and over in the years considered in the analysis. To create death rates, a denominator for each year, canton, age, and gender cell from intercensal interpolations of the population were used as an offset term.

The inequality measures and other canton-level independent covariates were constructed from the Costa Rican Household Surveys for Multiple Purposes (CRHS). Conducted in July of each year from 1976 until the present (except for 1984), the CRHS is nationally representative of households of approximately 1% of the population. From these surveys, the measure of total monthly household income was scaled by the square root of household size to account for intra-household sharing of assets (Foster, 2009) to create a household equivalent income measure. These income measures were then adjusted for inflation. Next, a

²Regressions not presented here show that areas with increasing mean incomes also have increasing Gini coefficients after accounting for canton fixed effects.

Gini coefficient was constructed based on this scaled income measure within cantons. These Gini measures were made by pooling two or three adjacent years of the micro data to ensure a sufficient number of households within each canton. The average number of observations used in creating each canton level Gini measures was 243 households. For certain years, the geocodes for the cantons were not available, so inequality measures were constructed only for years in which these codes could be ascertained from the data.³

These same household data and groupings were used to construct other canton-level variables including unemployment rate, in-migration, and mean equivalent household income. The unemployment rate was calculated using the CRHS and where unemployment was defined as those not working and looking for work in the last week. The in-migration measure captures the proportion of those surveyed who moved in the last two years and includes both internal and foreign migrants. The mean equivalent household income is the scaled monthly household income discussed above and the measure over which the Gini measure was calculated. Individual records from the CRHS were aggregated into cells defined by year and canton. The two data sources were combined based on year and canton, and the unit of analysis is canton, year, gender, and 5-year age group.

There are 81 cantons in Costa Rica, and they are the second hierarchical administrative division in Costa Rica. We choose to use administrative areas of cantons as our geographic unit of analysis because they are approximately analogous to a county, and have no independent taxation power (Sewastynowicz, 1983). According to 2000 Costa Rican census, cantons have an average population of about 50,000 and thus are on average about half the size of US counties.⁴

Statistical Analyses

Initially, we documented the progression of income values at different income percentiles across the study period at the national level.⁵ We also detailed canton-level income inequality and other canton-level measures descriptively to assess the values and changes over the study period.

We next estimated the association between changes in contemporaneous and 10 year lagged canton-level income inequality and changes in death rate by cause using multivariable Poisson models. The rates were created by using the number of deaths in each age, gender and canton cell as the dependent variable and accounting for population by using intercensal projections in each cell as an offset term in the Poisson models. In order to match population information with the counts of death, all the counts of death were combined for those aged 75 and over because the population data's final age category was 75 and over.

For each specific cause of death we estimate the following model (Model 1):

$$M_{ctag} = F_{\text{Poisson}}(\beta_0 + \beta_1(I_{ct}) + \beta_{2Z}(Z_{ct}) + \beta_3(I_{ct-10}) + \beta_{4Z}(Z_{ct-10}) + \beta_4 Y_t + \beta_5 A_a + \beta_6 G_g + \beta_7 A_a Y_t + \beta_8 \gamma_c + \varepsilon_{ctag})$$

where M_{ctag} is the mortality rate in canton c at year t for each gender g and 5-year age group a ; I_{ct} is the income inequality in canton c at time t , Z_{ct} is a vector of area-level controls; I_{ct-10} is the lagged income inequality in canton c , Z_{ct-10} is a vector of lagged area-level

³For 1987–1991 & 2005–2006, canton identifiers were encoded in the data. For 1995, 1996, 2001, and 2002, identifiers could be ascertained by other variables in the data. Consequently, the data was grouped as follows, 1987/1988, 1989/1990/1991, 1995/1996, 2005/2006.

⁴According to the US 2000 census, the average population of a US county was about 100,000.

⁵All incomes were adjusted to 2006 colones using consumer price index corrections (CPI). Details found at Banco Central De Costa Rica (<http://indicadoreseconomicos.bccr.fi.cr>).

controls; $\mathbf{Y}_t$ is a series of dummies for the years; $\mathbf{A}_a$ is a series of dummies for each age groups; $\mathbf{G}_g$ is a gender dummy; $\mathbf{A}_a\mathbf{Y}_t$ is a series of interactions of all age and year dummies to account for time varying improvements to mortality rates for age-specific groups that vary beyond the general time and age trends (Lee, 2000), and γ_c is a vector of canton specific fixed effect.⁶ Finally, the ε_{ctag} accounts for the remaining idiosyncratic error.

The main independent variables of interest are the constructed canton-level Gini measures, I_{ct-10} and I_{ct} . We also control for other lagged and contemporaneous canton-level time-varying characteristics, $\mathbf{Z}_{ct-10}$ and $\mathbf{Z}_{ct}$, including average household income, unemployment rate, and in-migration. These controls account for the local labor environment that may also be related to both inequality and mortality. For example selective migration patterns based on local economic opportunities may confound our estimates. For instance, increases in healthy migrant workers may increase both income inequality and at the same time decrease mortality rates in certain age groups. In addition to these controls, the fixed-effects further ensure that only changes in the income inequality variables are examined in relation to mortality outcomes.

We are particularly interested in the effects of lagged inequality because the impact of inequality on mortality would only be expected after a substantial time lag. While previous studies suggest a time lag of 10–15 years, we can only examine a time lag of 10 years repeatedly, so we chose this time lag for data reasons. With the canton-level fixed-effects, the coefficient on lagged inequality can be interpreted as the association of a change 10 years ago of income inequality on current changes in mortality. We also include contemporaneous inequality income to see if any association exists. If we find an association with contemporaneous inequality while controlling for lagged inequality, we interpret this association with caution because it is unlikely for inequality to affect health over the short-run. Nonetheless, we include both lagged and contemporaneous inequality income in the model simultaneously as has been suggested in a recent commentary (Glymour, 2008). In sensitivity analysis, we examine changes in the contemporaneous Gini separately from changes in lagged Gini measures.

Models are estimated separately for those aged 15–59 and those aged 60 and up because the literature suggests that SES effects are mitigated by age and effects of SES worse for working age individuals (Taylor et al., 1997). Robust standard errors are used to correct for heteroskedasticity and standard errors are clustered at the canton-level to correct for arbitrary serial correlation. We test for auto-regressive structures use clustered standard errors to adjust the standard errors because a recent study suggests that they perform better than AR type corrections (Bertrand et al., 2004). The estimated coefficients reflect a one-unit change in area income inequality on area-level mortality, yet this value is not directly interpretable because the Gini coefficient's entire range varies from 0–1. Consequently, the reported coefficients are transformed in terms of a 0.05 unit increase in the Gini coefficient for Tables 3 and 4. This is done so that the transformed coefficients approximately represent a 1 standard deviation change in the Gini coefficient. Finally, we employ Bonferroni multiple comparison corrections to account for the potential of type I error because of the large number of outcomes considered.

RESULTS

Figure 1 shows the secular progression of income and income distribution in Costa Rica over the study period at a national level. The percentage increases in monthly income at specific percentile thresholds are graphed over time. The percentage increases in mean

⁶Any variable that is bolded is meant to be a vector and the corresponding β is also a vector.

income at the 10th, 25th, 50th, 75th, 90th, and 99th percentile thresholds of the income distributions are presented. The household incomes, here after referred to as household equivalent mean income (HHEMI), adjust for household composition to account for household sharing of resources and were corrected for inflation. The graph shows that those in the low-income percentiles had very little growth in household equivalent income over this time, whereas those at the top of the income distribution enjoyed substantial growth. This figure shows that there was growing dispersion of income, and hence growing income inequality. The divergence is most clear after 1991 coinciding with years in which Costa Rica experienced significant GDP growth, which suggests that the rich were disproportionately favored in these growth years (A Rodríguez-Clare et al., 2002).

We next consider the regional variation in inequality. Costa Rica is comprised of 7 provinces that are subdivided into 81 cantons; these cantons are our geographic unit of analysis. Table 1 presents the summary statistics for the independent variables across cantons and time. Over this time period, the average Gini coefficient was generally increasing from 0.37 to 0.40. The most substantial changes in the Gini coefficients occurred during the 1990s, both nationally as is implied by Figure 1 and at the canton-level. Meanwhile, area-level HHEMI was relatively stable, but it exhibited increasing standard deviations over this time period. The other independent variables such as unemployment and in-migration rate were relatively stable over this period, although there were changes in unemployment during the 1990s.

Table 2 presents the results for Poisson regression analyses of the relations between canton-level inequality and all-cause mortality rates. The raw coefficients from the Poisson regressions are presented here. The first column presents the results for those aged 15–59, and the second presents the results for those aged 60 and over. Overall, neither changes in lagged nor changes in contemporaneous inequality relate to all-cause mortality for either age group. Of all the other canton-level covariates, surprisingly only lagged unemployment rate was associated with changes in the all-cause mortality rate for those aged 15–59. For the older group, none of the canton-level variables were significantly related to all-cause mortality rate.

Table 3 presents the incidence rate ratios (IRR) for lagged and contemporaneous change in income inequality and changes in rates of death for the 14 other causes examined for those aged 15–59. The comparable result for all-cause mortality is also presented. In this Table the IRRs have been scaled to reflect a 0.05 unit change in the cantons' Gini coefficients, which is comparable to a 1 SD change in the Gini coefficient. The results indicate that only liver disease mortality was significantly increased as lagged inequality increased. A one-standard deviation increase in the 10 year lagged Gini coefficient is associated with a 24% increase in the rate of liver disease mortality (IRR=1.24 CI=1.09–1.41). Mortality rates for two other causes, diabetes (IRR=1.18, CI=0.98–1.42) and suicide (IRR=1.13, CI=0.99–1.29), were also marginally associated with increases lagged income inequality. Moreover, results could not be estimated for two causes in this age group because there were too few observations.

Table 4 presents the incidence rate ratios for lagged and contemporaneous change in income inequality and changes in rates of death for the 14 causes considered for those 60 and over. Again, the comparable result for all-cause mortality is also presented, and again the IRRs have been scaled to reflect a 0.05 unit change in the Gini coefficient. Interestingly, changes in the lagged Gini coefficient were not significantly related to change in the rates of death for any of the causes considered. However, contemporaneous changes in the Gini coefficient were significantly related to decreases in the rates of breast cancer mortality (IRR=0.75, CI=0.92–0.61).

Given the large number of estimates presented in these analyses, we consider multiple test corrections to account for the potential of type I error. For each age group we estimated 15 regressions. Thus, we employ Bonferroni multiple comparison corrections, which require a significance threshold of 0.0033 for each test of the fifteen tests to recover an overall significance level of 0.05. Using this new significance cutoff, we find that increases in liver disease mortality remain significantly associated with increases in lagged inequality for those aged 15–59. For the older group, changes in breast cancer are no longer significantly associated with changes in contemporaneous income inequality using the new significance cut-off values.

Sensitivity Analysis

Our specification examines *changes* in the contemporaneous Gini measure and *changes* in the lagged Gini measure simultaneously. Given that we examine changes and not levels of income inequality, there is no *a priori* reason to believe that these *changes* are positively correlated, and in fact they may be negatively correlated because of mean reversion. However, to ensure that changes in the contemporaneous Gini do not mediate the pathway through which changes in lagged Gini measures affect mortality, we alter our specification. We rerun the basic model examining changes the contemporaneous Gini measures and changes in lagged Gini measures separately as a specification test.

This specification test confirmed the findings noted above. Again increases in 10 year lagged income inequality were associated with increases in mortality due to liver disease/cirrhosis for working aged individuals. The estimated coefficient was largely unchanged, and this result was robust to correction for multiple testing. In addition, the result suggesting that increases in 10 year lagged income inequality were associated with increases in mortality due the suicide for working aged individuals becomes slightly more significant (but was not robust to Bonferroni correction). Finally, we also do find that contemporaneous changes are related to decreases in breast cancer mortality for the elderly, but here the result becomes less significant (results in Appendix A & B).

DISCUSSION

Overall, we found some evidence that inequality is related to specific causes of mortality for working-aged adults, but no evidence of an all-cause relation. Increases in 10 year lagged income inequality were associated with increases in mortality due to liver disease/cirrhosis. This result was robust to correction for multiple testing. We also found that increases in 10 year lagged income inequality were associated with increases in diabetes and suicide for working age adults. These latter results were only marginally significant. However, they present a notable pattern in that the causes are directly related to health behaviors or perceptions of well-being.

We also note the lack of findings for chronic respiratory disease or other smoking related causes of death. We speculate that this may be due, at least in part, to the etiology and timing of diseases related to smoking. According to Doll et al., after 15 years of follow-up of 35 year olds there was a two-fold difference in mortality between life-long smokers and never smokers (and these differences are even more muted between former smokers and current smokers). However, these differences in mortality increased dramatically with longer follow-up periods (Doll et al., 2004). In contrast, Klatsky et al. found a 3.5 fold difference in cirrhosis mortality for moderate drinkers and 6.5–23 fold difference for heavy drinkers compared to abstainers and light drinkers (Klatsky et al., 1992) in only 10 years of follow-up. Thus, longer lags might be required to capture the timing correctly for smoking related diseases. Alternatively, smoking may not be one of the affected health behaviors.

In older adults, we find no relation with lagged inequality changes, but do find that contemporaneous changes are related to decreases in breast cancer mortality. This counterintuitive result may be related to access to secondary and tertiary health clinics that treat breast cancer, which may be more likely to be located in larger towns, where there is also more inequality (Brenes-Camacho and Rosero-Bixby, 2009). However, this result was not robust to correction for multiple testing.

Given our analysis approach, we believe the results support a psychosocial mechanism for income inequality despite modest effect sizes. We intentionally designed this analysis to err on the conservative side by only examining the variation in *changes* in lagged income inequality within canton in relation to *changes* in mortality rates. This was done to limit the potential for confounding (particularly by place) as much as possible; thus the small magnitude of our findings was not surprising. Our sensitivity analyses examining only lagged canton-level variables and our results were strengthened. We also conducted several other sensitivity analyses such as omitting cantons with dramatic changes in inequality and examining negative binomial models (not shown), and our results were robust to these specification tests. We also examine the results without canton fixed-effects. While the overall results still suggest no relationship between income inequality and mortality in Costa Rica, we find that our other area-level measures (such as in migration) become quite significant implying that these fixed effects are important to include.

Despite our careful analysis, there are several limitations in this study that merit attention. First, the inequality measures may be noisy as they are calculated over a small number of households for any given year. We expect this non-differential measurement error would make our results more attenuated. Second, the lagged measures do not correspond exactly to a 10-year lag because we only have inequality measures for certain years. In addition, the health consequences of inequality are not likely to work at the same rate for each disease and 10 years may only capture medium-term effects. We consider a 10 year lag for all causes examined for data reason, but disease specific time lags based on theoretical considerations would be best, as discussed above with smoking related diseases. Without such theoretical guidelines, we use a moderate time length and acknowledge its limitation in capturing all the potential effects. Third, to the extent that the study period spans a period of rapid economic development and trade liberalization, both inequality and health behaviors may be changing simultaneously, reflecting this more distal cause. While we mitigate some of this bias by looking at local changes in inequality, the processes may not be completely independent; thus suggesting that our results may reflect the transitional problems associated with development. Finally, the only individual level variables we were able to adjust in these models were age and sex (due to data availability) so while results are not confounded by basic demographics, there may be confounding by other individual characteristics, or by time variant factors associated with both changes in lagged inequality and mortality at the canton-level.

Notwithstanding these real limitations, this analysis of income inequality and mortality in the Costa Rican setting does provide some insight into reasons for the relation between income inequality and health and suggests a mediating role for health behaviors. The results from this setting, where we are less concerned with discrimination and political confounding, echo results from other settings where health related behaviors were related to inequality (Galea et al., 2003; Miller et al., 2005). It seems that inequality does “get under one’s skin” for the Costa Rican working age population through changes in behaviors such as alcohol consumption, which is related to cirrhosis and liver disease. In addition, we also found that changes in lagged income inequality relate to causes of death related to diet (diabetes), and life-satisfaction (suicide). While these latter results were only marginally significant, they are consistent with a behaviorally mediated pathway. There is growing

evidence that health behaviors, as opposed to underlying biological dysregulation, mediate the overall SES and health relationship, which is congruent with our findings here (Dowd and Goldman, 2006; Stringhini et al., 2010). More research in this setting is required to understand how and if health behaviors changed during this time period.

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JHAP 1056 -Research Highlights

- We examined the longitudinal relation between income inequality and mortality in Costa Rica
- Inequality was not related to all-cause mortality
- Inequality was related to liver disease mortality for those aged 15–60
- Inequality was marginally related to diabetes and suicide mortality for those 15–60
- Income inequality may impact the health behavior of Costa Ricans of working age

Figure 1.

Percentage change in household equivalent income from 1987 to 2005 by income percentile. Source: Author's calculation based on Costa Rican Household Survey for Multiple Purposes and Costa Rican CPI deflators found at Banco Central De Costa Rica (<http://indicadoreseconomicos.bccr.fi.cr>). All colones adjusted to 2006 colones.

Table 1

Descriptive statistics for canton-level covariates.

	Canton Characteristics		
	Year	Mean	Std. Dev.
Gini Coefficient	2005	0.391	0.052
	2001	0.414	0.061
	1995	0.373	0.049
	1990	0.374	0.048
	1987	0.382	0.058
(Mean HHEMI)/1000 in 2006 Colones	2005	173.93	61.83
	2001	183.77	69.50
	1995	152.85	50.81
	1990	160.93	46.77
	1987	172.09	55.24
Migration	2005	0.046	0.025
	2001	0.040	0.024
	1995	0.045	0.023
	1990	0.038	0.021
	1987	0.061	0.040
Unemployment Rate	2005	0.062	0.019
	2001	0.067	0.027
	1995	0.057	0.022
	1990	0.047	0.019
	1987	0.055	0.026
Observations	77		

Source: Author's calculation based on Costa Rican Household Survey for Multiple Purposes and Costa Rican CPI deflators found at Banco Central De Costa Rica (<http://indicadoreseconomicos.bccr.fi.cr>). All income values adjusted to 2006 colones. 77 of 81 cantons are included

Table 2

Association of contemporaneous and lagged canton characteristics and all-cause mortality by age group.

	All- Cause Mortality	
	Aged 15–59	Aged 60+
Δ Contemporaneous Area Variables		
(Mean HHEMI)/100,000	−0.00140 (0.00604)	−0.00154 (0.004330)
Gini Coefficient	0.1828 (0.3865)	−0.2571 (0.2693)
Unemployment Rate	0.4313 (0.8538)	0.3516 (0.4325)
Migration	−0.2954 (0.7495)	−0.4326 (0.4328)
Δ Lagged Area Variables		
(Mean HHEMI)/100,000 (T-10)	0.001959 (0.008511)	0.001367 (0.004669)
Gini Coefficient (T-10)	0.3126 (0.4183)	−0.4922 (0.2631)
Unemployment Rate (T-10)	−2.0683* (0.9304)	0.6864 (0.5309)
Migration (T-10)	0.9478 (0.5474)	−0.06085 (0.3198)
Constant	−7.5759** (0.2712)	−4.2829** (0.1411)
Canton Fixed Effects	Yes	Yes
Observations	4158	1848

Robust and clustered standard errors in parentheses

* significant at 5%;

** significant at 1%

Other controls include age (5-year brackets), gender, year fixed-effects, age & year indicator interactions. The unit of observation is canton, year, gender, 5-year age group. Population in age/gender/canton cell in corresponding year was used as an offset.

Table 3

Comparison of incident relative risk (IRR) of mortality by cause as estimated for 0.05 unit change in the contemporaneous and lagged inequality canton Gini coefficients. Ages 15–59. N=4158 for each regression.

	Age 15–60			
	Δ Contemporaneous Gini		Δ Lagged Gini	
	IRR	95% CI	IRR	95% CI
All-Cause	1.003	(0.969–1.039)	1.016	(0.978–1.057)
Tuberculosis	0.914	(0.488–1.712)	1.192	(0.756–1.879)
Acute Respiratory (Infection)	1.073	(0.823–1.399)	0.945	(0.742–1.202)
Cancer of Digestive System	0.942	(0.824–1.077)	1.116	(0.924–1.348)
Cancer of Respiratory System	0.809	(0.571–1.148)	0.893	(0.623–1.278)
Cervical Cancer	Did Not Converge			
Breast Cancer	1.022	(0.838–1.248)	1.055	(0.823–1.352)
Prostate Cancer	Did Not Converge			
Chronic Respiratory Causes	0.925	(0.771–1.108)	0.896	(0.721–1.113)
Cardio Vascular Disease	0.988	(0.922–1.058)	0.927	(0.855–1.005)
Diabetes	1.046	(0.883–1.239)	1.182	(0.982–1.422)
Alcohol/Liver Disease	1.075	(0.949–1.217)	1.239	(1.088–1.412)** <i>B</i>
Motor Vehicle Accident	0.999	(0.907–1.1)	1.018	(0.893–1.162)
Suicide	1.061	(0.92–1.223)	1.130	(0.988–1.292)
Homicide	0.918	(0.811–1.039)	1.003	(0.873–1.153)

Robust and clustered standard errors in parentheses

* significant at 5%;

** significant at 1%

B Significant at the 0.05 level after multiple comparison correction. (The Bonferroni multiple comparison correction for fifteen tests of significance requires a significance threshold of 0.0033 for each test to recover an overall significance level of =0.05)

Area controls include mean household equivalent monthly income, unemployment rate, percent in-migration, lagged mean household equivalent monthly income, lagged unemployment rate, and lagged percent in-migration. Other controls include age (5-year brackets), gender, year fixed-effects, canton fixed-effects, and age & year indicator interactions. The unit of observation is canton, year, gender, 5-year age group. Population in age/gender/canton cell in corresponding year was used as an offset.

Two causes did not converge because there were too few deaths from these causes in this age group.

Table 4

Comparison of incident relative risk (IRR) of mortality by cause as estimated for 0.05 unit change in the contemporaneous and lagged inequality canton Gini coefficients. Ages 60+. N=1848 for each regression.

	Age 60+			
	Δ Contemporaneous Gini		Δ Lagged Gini	
	IRR	95% CI	IRR	95% CI
All-Cause	0.984	(0.96–1.009)	0.976	(0.951–1.001)
Tuberculosis	1.088	(0.826–1.433)	1.026	(0.708–1.486)
Acute Respiratory (Infection)	0.948	(0.831–1.081)	0.976	(0.852–1.117)
Cancer of Digestive System	0.953	(0.875–1.037)	0.978	(0.889–1.076)
Cancer of Respiratory System	0.929	(0.808–1.067)	1.002	(0.878–1.143)
Cervical Cancer	1.043	(0.824–1.321)	1.070	(0.847–1.352)
Breast Cancer	0.743	(0.609–0.907)**	1.039	(0.839–1.286)
Prostate Cancer	1.028	(0.88–1.202)	0.951	(0.824–1.098)
Chronic Respiratory Causes	0.993	(0.933–1.056)	0.993	(0.929–1.062)
Cardio Vascular Disease	1.016	(0.977–1.056)	0.979	(0.941–1.018)
Diabetes	0.975	(0.899–1.058)	0.920	(0.84–1.007)
Alcohol/Liver Disease	0.956	(0.837–1.092)	0.919	(0.787–1.073)
Motor Vehicle Accident	1.007	(0.839–1.208)	0.814	(0.65–1.019)
Suicide	0.893	(0.557–1.431)	1.305	(0.812–2.099)
Homicide	0.985	(0.748–1.298)	1.127	(0.864–1.47)

Robust and clustered standard errors in parentheses

* significant at 5%;

** significant at 1%

^B Significant at the 0.05 level after multiple comparison correction. (The Bonferroni multiple comparison correction for fifteen tests of significance requires a significance threshold of 0.0033 for each test to recover an overall significance level of =0.05)

Area controls include mean household equivalent monthly income, unemployment rate, percent in-migration, lagged mean household equivalent monthly income, lagged unemployment rate, and lagged percent in-migration. Other controls include age (5-year brackets), gender, year fixed-effects, canton fixed-effects, and age & year indicator interactions. The unit of observation is canton, year, gender, 5-year age group. Population in age/gender/canton cell in corresponding year was used as an offset.

Appendix A

Comparison of incident relative risk (IRR) of mortality by cause. Models run separately for changes in the contemporaneous and lagged canton Gini coefficients. Presented IRR are for a 0.05 unit change in the respective Gini coefficient. Ages 15–59. 30 regressions summarized in this table with N=4158 for each regression.

	Age 15–60			
	Model A: Δ Contemporaneous Gini Only		Model B: Δ Lagged Gini Only	
	IRR	95% CI	IRR	95% CI
All-Cause Mortality	0.998	(0.965–1.033)	1.017	(0.977–1.059)
Tuberculosis	0.967	(0.591–1.582)	1.139	(0.694–1.871)
Acute Respiratory (Infection)	1.062	(0.848–1.329)	0.944	(0.75–1.187)
Cancer of Digestive System	0.922	(0.814–1.043)	1.133	(0.932–1.377)
Cancer of Respiratory System	0.739	(0.536–1.018)	0.926	(0.643–1.336)
Cervical Cancer				
Breast Cancer	0.996	(0.806–1.231)	1.076	(0.841–1.378)
Prostate Cancer				
Chronic Respiratory Causes	0.911	(0.769–1.081)	0.929	(0.744–1.16)
Cardio Vascular Disease	0.978	(0.917–1.044)	0.924	(0.852–1.001)
Diabetes	0.990	(0.826–1.187)	1.190	(0.998–1.419)
Alcohol/Liver Disease	1.034	(0.926–1.155)	1.244	(1.096–1.412) ^{**B}
Motor Vehicle Accident	1.005	(0.911–1.11)	1.017	(0.89–1.162)
Suicide	1.021	(0.895–1.166)	1.147	(1.012–1.299) [*]
Homicide	0.939	(0.832–1.06)	0.998	(0.866–1.149)

Robust and clustered standard errors in parentheses

* significant at 5%;

** significant at 1%

^B Significant at the 0.05 level after multiple comparison correction. (The Bonferroni multiple comparison correction for fifteen tests of significance requires a significance threshold of 0.0033 for each test to recover an overall significance level of =0.05)

Area controls include mean household equivalent monthly income, unemployment rate, and percent in-migration for the contemporaneous model presented in the first two columns. Area controls include lagged mean household equivalent monthly income, lagged unemployment rate, and lagged percent in-migration for the lagged model presented in the last two columns. Both models include other controls include age (5-year brackets), gender, year fixed-effects, canton fixed-effects, and age & year indicator interactions. The unit of observation for both models is canton, year, gender, 5-year age group. Population in age/gender/canton cell in corresponding year was used as an offset. Two causes did not converge because there were too few deaths from these causes in this age group.

Appendix B

Comparison of incident relative risk (IRR) of mortality by cause. Models run separately for changes in the contemporaneous and lagged canton Gini coefficients. Presented IRR are for a 0.05 unit change in the respective Gini coefficient. Ages 60+. 30 regressions summarized in this table with N=1848 for each regression.

	Age 60+			
	Model A: Δ Contemporaneous Gini Only		Model B: Δ Lagged Gini Only	
	IRR	95% CI	IRR	95% CI
All-Cause Mortality	0.988	(0.965–1.012)	0.978	(0.953–1.004)
Tuberculosis	1.056	(0.813–1.37)	1.006	(0.689–1.469)
Acute Respiratory (Infection)	0.949	(0.84–1.072)	0.982	(0.856–1.127)
Cancer of Digestive System	0.955	(0.886–1.03)	0.984	(0.892–1.084)
Cancer of Respiratory System	0.929	(0.815–1.06)	0.998	(0.881–1.132)
Cervical Cancer	1.072	(0.857–1.34)	1.068	(0.855–1.335)
Breast Cancer	0.789	(0.646–0.963)*	1.023	(0.829–1.263)
Prostate Cancer	0.989	(0.846–1.155)	0.960	(0.833–1.106)
Chronic Respiratory Causes	0.995	(0.948–1.044)	0.995	(0.93–1.064)
Cardio Vascular Disease	1.016	(0.978–1.056)	0.982	(0.947–1.02)
Diabetes	0.949	(0.884–1.02)	0.924	(0.842–1.015)
Alcohol/Liver Disease	0.948	(0.84–1.069)	0.927	(0.788–1.09)
Motor Vehicle Accident	1.019	(0.852–1.219)	0.812	(0.64–1.029)
Suicide	1.029	(0.702–1.508)	1.295	(0.798–2.104)
Homicide	1.015	(0.753–1.369)	1.120	(0.857–1.465)

Robust and clustered standard errors in parentheses

* significant at 5%;

** significant at 1%

^B Significant at the 0.05 level after multiple comparison correction. (The Bonferroni multiple comparison correction for fifteen tests of significance requires a significance threshold of 0.0033 for each test to recover an overall significance level of =0.05)

Area controls include mean household equivalent monthly income, unemployment rate, and percent in-migration for the contemporaneous model presented in the first two columns. Area controls include lagged mean household equivalent monthly income, lagged unemployment rate, and lagged percent in-migration for the lagged model presented in the last two columns. Both models include other controls include age (5-year brackets), gender, year fixed-effects, canton fixed-effects, and age & year indicator interactions. The unit of observation for both models is canton, year, gender, 5-year age group. Population in age/gender/canton cell in corresponding year was used as an offset.